ernment agencies ruling upon what can or cannot be prescribed for what purpose, which operations are permissible under what circumstances and which are not, how long a patient may remain in a given type of health care facility with a given diagnosis and how many hours of formal continuing education a physician must have to keep his license to practice?

Would it not be much better—and much cheaper—for government and social policy to find ways to encourage and strengthen the motivation and conscience of the physician, rather than to force him into a defensive attitude where he must look upon both his patients and his government as potential enemies? After all, the attitude, motivation and conscience of physicians are essential, if not critical ingredients of both the cost and the quality of patient care. So far there is little evidence that government controls have done that much either to reduce costs or to improve quality. Maybe it is time for a more sophisticated approach.

—MSMW

Pulmonary Vascular Disease

PULMONARY EMBOLISM is a common problem often complicating the course of patients in hospital. The exact incidence is uncertain, but some studies suggest that the diagnosis may be missed in as many as 60 percent of patients dying on a general medical service. The natural history of pulmonary embolism is also uncertain, but the reported recurrence rate is as high as 20 to 40 percent in the first six months after a clinically-evident embolism. Does this recurrence rate decrease if the risk factor (such as congestive failure, contraceptive pills or long-bone factures) is eliminated? The answer to this question is critical to proper management, but data from appropriately controlled, prospective trials are not yet available.

Recurrent pulmonary embolism may present, often in relatively young, active persons, as dyspnea of unknown origin. This interesting, perplexing and theoretically preventable syndrome is elegantly reviewed in the Medical Staff Conference in this issue of THE WESTERN JOURNAL OF MEDICINE. The correct diagnosis depends on a high index of suspicion in the attending physician. If a patient has complaint of effort dyspnea (often episodic initially) that has caused a significant change of life style, a diagnosis of pulmonary embolism should be considered—particularly if none of the usual cardiopulmonary causes of dyspnea are present. Physicians should avoid attributing the dyspnea to psychoneurosis. In many patients psychoneurotic symptoms develop after being told repeatedly that "it's all in your head." Moreover, evidence of death of lung tissue, pulmonary hypertension, right ventricular enlargement and, finally, failure are all very late manifestations of this illness. A physician must not wait for these abnormalities to appear to confirm his suspicion.

Few physiological abnormalities may be present at rest, but an arterial blood sample usually will show evidence of chronic hyperventilation—decreased carbon dioxide partial pressure (PCO_2), decreased bicarbonate (HCO_3) and relatively normal hydrogen ion concentration (pH). This is a useful objective sign that the patient may have an abnormal ventilatory drive often associated with pulmonary vascular obstruction. Furthermore, although redistribution of blood flow may result in preservation of a normal carbon monoxide uptake, this is not the rule; in a series of 31 patients with documented chronic pulmonary vascular obstruction studied in our laboratory, diffusing capacity of the lung for carbon monoxide (DLCO) was decreased below 80 percent of predicted in 23 (mean DLCO: 11.8 ml/min X mmHg). DLCO can be measured quickly and noninvasively so it can be useful in screening patients in whom this diagnosis is suspected.

However, in most cases it will be necessary to evaluate these patients during exercise. During exercise, alveolar hyperventilation usually persists, arterial hypoxemia often develops, DLCO does not increase normally, while wasted ventilation increases to grossly abnormal levels, as more and more ventilation goes to poorly perfused alveoli. Pulmonary artery pressure increases to abnormal levels also, compared to that in healthy persons of comparable age, particularly when related to pulmonary blood flow.

The cause of the breathlessness in these unfortunate patients is unknown. It may be due to receptors in the walls of precapillary vessels which detect their abnormal distensibility, especially dur-

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ing exercise when pulmonary arterial pulse pressure increases. Whatever the mechanism, experiments in animals and humans suggest that abnormal afferent signals are triggered in the vagus nerves by pulmonary vascular lesions. Mills and co-workers[1] reported that pulmonary embolism in rabbits increased firing rates in rapidly-adapting receptors innervated by single vagal nerve fibers. Gus and co-workers[2] showed that unilateral vagal blockade in patients with pulmonary vascular disease was associated with a decrease in hyperventilation and relief of dyspnea. If confirmed, these studies suggest possible therapeutic approaches to relieve dyspnea in such patients.

Future studies should clarify the natural history of the disease, particularly with respect to correction of risk factors; improve our methods of diagnosing occult venous disease as well as pulmonary embolism itself; and improve therapy, prophylactic as well as corrective.

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